Group A β-Hemolytic Streptococcal Toxic Shock from a Mild Pharyngitis

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The advent of antibiotics and the improvement in socioeconomic conditions have brought about a decline in virulent strains of group A β-hemolytic streptococcus (GABHS) and therefore the complication rate.[1] Recent studies, however, suggest a reversal of this trend. In one report, 20 patients from the Rocky Mountain region became ill (1986 to 1988) with a GABHS toxic shock syndrome.[2] There was a 30% mortality rate. Seven had preexisting diseases identified, and in only two was the pharynx the portal of entry.

Other reports associating GABHS with sepsis have linked most of the patients with underlying illnesses or predisposing factors.[3–6] These preexisting conditions include malignancy, immunosuppressed states, diabetes mellitus, alcoholism, surgeries, trauma, foreign body, nosocomial infections, intravenous drug abuse, prior illness (ie, varicella), and neurologic disorders. Mortality rates have been high, and they increase in the presence of shock.[6] What is now called streptococcal toxic shock syndrome[7–9] is secondary to the production of a pyogenic exotoxin[10] producing vascular collapse. Reported herein is the case of an 18-year-old female Floridian, in good health, who in 1987 developed GABHS septic shock preceded by a mild pharyngitis. She was successfully treated, and a 3-year follow-up period has not uncovered any underlying diseases or risk factors.

CASE REPORT

A healthy 18-year-old woman presented in November 1987, with a 1-week history of initially mild sore throat and headache followed by fever, chills, sweats, vomiting, diarrhea, fainting spells, and a painful swelling on the right side of her neck. The sore throat, recalled only after repeated questioning, had resolved 3 days earlier and was described as only a mild scratchiness. She reported no cough, no chest, abdominal, or back pain, and no urinary symptoms, but she did complain of a severe headache. Her only medications were birth control pills, and her last normal menstrual period finished 1 week before presentation. Tampons, used during menses, had not been used since then. There was no history of urinary tract infection, diabetes mellitus, malignancies, drug or alcohol abuse, exposure to sexually transmitted diseases, the human immunodeficiency virus (HIV-I), tuberculosis, or streptococcal infections.

On examination the patient was found to be toxic, diaphoretic, and dehydrated. Reclining blood pressure was 90/60 mm Hg with a pulse of 120 beats per minute, which changed to 80/60 mm Hg and 140 beats per minute, on sitting up. Oral temperature was 38.6°C (101.5°F), and her respiratory rate was 20/min. There was a mildly erythematous pharynx without exudates. The neck was supple without cervical adenopathy, but an ill-defined, very tender mass at the right base of the neck extended into the supraclavicular fossa. Lungs were clear, there were no cardiac murmurs, the abdomen was benign without hepatosplenomegaly, and a questionable right-sided costovertebral angle tenderness was noted. There were no rashes, stigmata of endocarditis, or lymphadenopathy present, and the patient was neurologically intact. Admission white cell count was 16.9×10^9/L (16,900/mm^3) with 0.61 segmented neutrophils, 0.36 banded neutrophils and 0.3 lymphocytes. Urinalysis was remarkable for proteinuria (3+), a few bacteria, and 5 to 10 white blood cells per high-powered field. Serum chemistry determinations revealed prerenal azotemia and hepatopathy by elevated liver enzymes and prolonged prothrombin time. Electrolytes were normal, and the blood glucose was 6.8 mmol/L (124 mg/dL). A test for infectious mononucleosis (Monospot) was negative. The patient's chest x-ray examination was normal.

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The patient was hospitalized with a diagnosis of septic shock, tentatively thought to be from pyelonephritis, and unexplained neck mass. Because of the headache, a lumbar puncture and computed tomographic (CT) scan of the head were performed, the findings of which were unremarkable. Blood, urine, throat, cerebral spinal fluid, and stool cultures were obtained, and the patient was treated with intravenous hydration, cefotaxime, and nafcillin. A CT scan of the neck and the chest, performed the next day, was compatible with cellulitis of the neck without soft tissue gas or retropharyngeal abscesses. A moderate-sized right-sided pleural effusion was noted. A sterile transudate was documented by thoracentesis. Blood and throat cultures grew GABHS at 24 hours. All other cultures were negative, including an acid-fast bacillus stain of pleural fluid. The antibiotic was switched to intravenous aqueous penicillin G, 1 million units every 4 hours.

Other tests included a tuberculosis skin test, HIV-I antibody test, antinuclear antibody test, rheumatoid factor, hepatitis A and B profiles, protein and immunoelectrophoresis, and immunoglobulin G subclasses. All were unrevealing. An antistreptolysin O titer, determined on the second hospital day, was not elevated, perhaps because of early antibiotic therapy and brief duration of illness.

The patient became afebrile on the 3rd hospital day and clinically showed dramatic improvement. On the 7th hospital day, a cardiac murmur was detected on auscultation. Mitral valve prolapse with regurgitation was diagnosed by echocardiogram, and there were no vegetations present. The patient was treated with intravenous penicillin G, 1 to 2 million units for a total of 14 days, and the neck swelling resolved. The infectious disease consultant concurred with 2 rather than 4 weeks of intravenous antibiotics because there was no clinical evidence of endocarditis or valvular vegetations found by echocardiography. Liver and renal function tests returned to normal. She was discharged with a 2-week prescription of oral penicillin, 500 mg 4 times a day, and after a follow-up period of 3 years, she has remained in good health.

DISCUSSION

This patient's clinical picture is consistent with postanginal sepsis, a severe and rare complication of pharyngitis.[11] It sometimes appears even after an insignificant sore throat. Various aerobic or anaerobic bacteria can be responsible for the septicemia. In this case, GABHS was isolated from the blood and pharynx. The neck swelling is consistent with thrombophlebitis of the internal jugular vein with associated cellulitis of the cervical soft tissues. Infectious foci are embolized into the vascular system and disseminate. The patient then rapidly deteriorates into shock. Alternatively, cervical fasciitis with cellulitis could explain this patient's neck mass. The pleural effusion suggests pulmonary involvement, but more likely it represents serous excretions into the pleural space from inflamed soft tissues at the root of the neck. A contrasting feature to some prior reports is this patient's underlying good health. Young healthy adult patients presenting with fever and shock are more likely to have gram-negative septicemia originating from the genitourinary or gastrointestinal tracts. In this patient, bacteriologic investigation identified the source. Staphylococcal toxic shock syndrome is ruled out because the patient did not have the triad of fever, hypotension, and rash, and had positive cultures for GABHS.[12]

Harden and Lennon[13] have identified 13 previously well children in New Zealand with bacteremia from serious suppurative group A streptococcal infections. Most cases were in infants or young children, and none was older than 10 years of age. Also, long-term follow-up for the whole group was not discussed. Hess and Grant[14] reported a 19-year-old woman who died of complications from GABHS pharyngitis, and who only after autopsy was discovered to have disseminated malignant histiocytosis. In the series reported by Stevens et al,[2] it is not known whether the otherwise healthy patients have remained that way. Additionally, their patients resided in the Rocky Mountain states. In 1989, three patients from the Middle Atlantic region with GABHS toxic shock syndrome were reported.[15] This patient's illness confirms the presence of virulent strains of GABHS in the southeastern United States about the same time that it was observed elsewhere. The reader is alerted to this changing epidemiology of GABHS.

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